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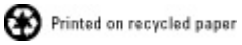

ABSTRACT

The U.S. Environmental Protection Agency (EPA) has announced plans to regulate mercury (Hg) emissions from coal-fired power plants. EPA has not prepared a quantitative assessment of the reduction in risk that could be achieved through reduction in coal plant emissions of Hg. To address this issue, Brookhaven National Laboratory (BNL) with support from the U.S. Department of Energy Office of Fossil Energy (DOE FE) prepared a quantitative assessment of the reduction in human health risk that could be achieved through reduction in coal plant emissions of Hg. The primary pathway for Hg exposure is through consumption of fish. The most susceptible population to Hg exposure is the fetus. Therefore the risk assessment focused on consumption of fish by women of child-bearing age. Dose response factors were generated from studies on loss of cognitive abilities (language skills, motor skills, etc.) by young children whose mothers consumed large amounts of fish with high Hg levels. Population risks were estimated for the general population in three regions of the country, (the Midwest, Northeast, and Southeast) that were identified by EPA as being heavily impacted by coal emissions. Three scenarios for reducing Hg emissions from coal plants were considered: (1) A base case using current conditions; (2) A 50% reduction; and, (3) A 90% reduction. These reductions in emissions were assumed to translate linearly into a reduction in fish Hg levels of 8.6% and 15.5%, respectively. Population risk estimates were also calculated for two subsistence fisher populations. These groups of people consume substantially more fish than the general public and, depending on location, the fish may contain higher Hg levels than average. Risk estimates for these groups were calculated for the three Hg levels used for the general population analyses. Analysis shows that the general population risks for exposure of the fetus to Hg are small. Estimated risks under current conditions (i.e., no specific Hg controls) ranged from 5.7×10^{-6} in the Midwest to 2×10^{-5} in the Southeast. Reducing emissions from coal plants by 90% reduced the estimated range in risk to 5×10^{-6} in the Midwest and 1.5×10^{-5} in Southeast, respectively. The population risk for the subsistence fisher using the Southeast regional fish Hg levels was 3.8×10^{-3}, a factor of 200 greater than the general population risk. For the subsistence fishers and the Savannah River Hg levels, the population risk was 4.3×10^{-5}, a factor of 2 greater than for the general population. The estimated risk reductions from a 90% reduction in coal plant Hg emissions ranged from 25% – 68%, which is greater than the assumed reduction in Hg levels in fish, (15.5%). To place this risk in perspective, there are approximately 4×10^{6} births/year in the U.S (National Vital Statistics Report, 2000). Assuming that the Southeast risk level (the highest of the regions) is appropriate for the entire U.S., an estimate of 80 newborn children per year have a 5% chance of realizing any of the 16 adverse effects used to generate the DRF. If Hg emissions from power plants are reduced 90%, the number of children at risk is reduced to 60.

1. INTRODUCTION

Mercury (Hg) contamination in fish is a perceived concern in the United States and many countries of the world. Forty-one states have fish consumption advisories due to Hg contamination. Hg is a trace impurity in coal and a fraction of this Hg is released to the atmosphere during combustion. U.S. coal-fired power plants constitute the largest point source of anthropogenic Hg contributing approximately one-third of the anthropogenic Hg released in the U.S.

Although the U.S. Environmental Protection Agency (EPA) has announced its intention to control Hg emissions from coal-fired power plants, EPA has not prepared a quantitative assessment of the reduction in risk that could be achieved through reduction in coal plant emissions of Hg. To address this issue, Brookhaven National Laboratory (BNL) with support from the U.S. Department of Energy Office of Fossil Energy (DOE FE) prepared a risk assessment of Hg exposure from fish consumption for three regions of the U.S. (Northeast, Southeast, and Midwest) identified by the EPA as regions to be more highly impacted by Hg releases from coal-fired power plants. This risk assessment addresses the effects of exposure to *in utero* children through consumption of fish by their mothers. Two population groups (general population and subsistence fishers) are considered. Three Hg levels are considered in the analysis, current conditions based on measured data, and hypothetical reductions in Hg levels due to a 50% and 90% reduction in Hg emissions from coal-fired power plants. This report presents the results of this analysis and updates previous assessments of the health risks of methylmercury that BNL prepared for DOE FE. A comprehensive report was prepared (Lipfert et al., 1994) that led to several journal articles and conference presentations (Lipfert et al. 1994, 1995, 1996).

Since that original effort, important governmental publications have appeared (EPA, 1997; NRC, 2000) and new information has been published in the literature including, but not limited to:

- Hg emissions have been estimated for each individual coal power plant in the United States and data on Hg emissions by species have been obtained.
- Data on Hg control technologies, their costs and ability to remove Hg have been refined.
- More sophisticated air dispersion modeling has been performed, including estimates of global contributions to Hg deposition in the U.S.
- Measurements of wet Hg deposition and air concentrations have been made at a number of locations in the U.S.
- More data have been obtained on Hg in fish, by state and region.
- More data have been obtained on human exposure to Hg, including differences due to fish consumption.

- More results from major epidemiological studies have been published, including those of the Seychelles and Faeroe Islands and of Amazon Indians.

This information provides improved data for the risk assessment prepared by BNL. As background material, this report discusses the Hg cycle in the atmosphere and through aquatic food chains. This material covers some of the complexities that introduce uncertainties in performing a risk assessment. Based on the Hg cycle, a simplified approach to calculating risks is presented. The approach is based on three major topics; MHg emissions and deposition (emphasizing coal plants), Hg consumption through fish, and dose response functions for Hg. Each of these topics is covered in the report. Using this information a quantitative risk assessment is prepared and the impact of reducing Hg emissions from coal-fired power plants is assessed. Based on the findings recommendations for future work and conclusions are provided.

2. MERCURY CYCLE

Hg is released to the atmosphere from both natural and anthropogenic sources. Natural sources include re-emission from vegetative plants and water bodies, as well as spatially discrete larger-scale events such as volcanic activity or forest fires. Anthropogenic sources include coal combustion plants, waste incinerators, volatilization from paints, fungicides and other Hg containing products, smelting, and chloralkali plants. There are three major forms of gaseous Hg, elemental mercury Hg(0), reactive gaseous mercury Hg^{+2}, and particulate mercury Hg(p). Elemental Hg is the predominant form in the atmosphere and it persists in the atmosphere for approximately one year before being deposited. Approximately 1 – 3% of the Hg in the atmosphere is Hg^{+2} and a smaller percentage is particulate Hg. Hg^{+2} and Hg(p) are transported much shorter distances than elemental Hg prior to deposition. These two forms of Hg are deposited primarily through rainfall, however dry deposition also occurs.

Some deposited Hg will find its way to water bodies. There Hg accumulates in vegetation in the water. These plants are consumed by small fish, which are consumed by larger fish. At each stage, Hg concentrations increase (e.g. bioaccumulation occurs). At the highest trophic level, the Hg concentration in the fish can be millions of times larger than in the water column. Hg concentrations in fish often reach the ppm level. Fish advisories recommending reduced consumption are typically provided when Hg concentrations are around 1 ppm. Consumption of fish has been identified as the major pathway for accumulation of Hg in humans.

Although the general cycle is well understood the exact details are not. There are still large uncertainties in a number of areas that impact the risk assessment. These include:

- Effects of point sources (e.g. coal power plants) on local deposition.
- Effects of anthropogenic global sources on deposition in the U.S.
- Effects of deposition on Hg loadings in water bodies.
- Effects of Hg loadings in water bodies to concentrations in fish.

- Methylation process that converts Hg to a form that accumulates in fish.

In addition, there is large uncertainty in the response of the environment to reduced Hg emissions. Over 90% of the Hg emitted since the start of the industrial revolution is contained in water bodies, sediments and soil. A reduction in Hg emissions would most likely be buffered through releases from these other sources. Expert panels have estimated that it would take 15 – 25 years before the impacts of reduction in Hg emissions could be observed. (Minnesota, 1999, USEPA, 1998b).

3. RISK ASSESSMENT APPROACH

EPA has acknowledged that most of the population is not at risk from Hg contained in fish. For most of the population, eating fish is recommended because of the many health benefits that it provides, in spite of concerns about Hg. The population at greatest risk is the *in utero* child. For this reason, this risk assessment is focused on women of child bearing age (16 – 49); especially "fishers (aka subsistence fisherman)" or individuals who eat an unusually large amount of fish.

The end-point used in this study is the population risk of a specified health effect which is estimated as the sum of the products of the incremental probability of exposure at a given level for each member of the population times the probability of experiencing the effect at that exposure level. Information on such responses is obtained from a "dose-response" function, where some measure of individual exposure serves as a proxy for the dose to the target organ, here the developing fetal brain.

This paradigm requires data on the distribution of exposures (either measured or calculated) and a dose-response function, both expressed in terms of the same exposure metric. Human Hg exposures are expressed as concentrations in hair for this study. Other measures of exposure (biomarkers) are Hg concentrations in blood and umbilical cords.

The baseline risk assessment approach has the following steps:

- Estimate fish consumption by species from survey data.
- Estimate Hg concentration in fish species from measured data.
- Estimate daily intake as the product of consumption and concentration in fish.
- Convert intake into levels of Hg in hair.
- Use the dose response function to estimate risk.

Each member of the population must be examined to get the population risk. In fact, the consumption of fish varies from person to person and the Hg concentration in fish varies

between fish and between species of fish. Therefore, to get the population risk, a Monte Carlo approach that samples among the distribution of consumption behavior (e.g., fishers vs. the general population) and the distribution of Hg concentrations in fish is used. The result is a distribution of daily intake (i.e. 3% of the population has an intake of 1 ug/d, 5% has an intake of 2 ug/d, and so on). This distribution in intake is converted to a distribution in Hg in hair. The dose response function is used for each group and the results are summed to estimate the total population risk.

To examine the impacts of reducing Hg emissions from coal plants, the following additional steps are required:

- Estimate the reduction in Hg emissions.
- Assume that a reduction in emissions corresponds to the same reduction in mean Hg levels in fish.
- Using the reduced mean Hg levels, repeat the baseline process.

As a comparison, the predicted distribution of Hg levels in hair from the baseline case and the reduced emissions case is used as well as the change in population risk. Using the above approach involves a number of assumptions that implies that there are uncertainties in the analysis. To provide context to these uncertainties, they will be discussed after the completion of the quantitative risk assessment.

The next few sections provide the data and technical basis for the risk assessment. This includes discussions on Hg emissions and potential reductions from coal-fired plants, fish consumption, Hg levels in fish, data on Hg levels in humans, and estimates of possible dose response functions. This is followed by the assessment of the impact of reducing Hg emissions on human health risk.

4. MERCURY EMISSIONS AND DEPOSITION FROM COAL PLANTS

In 1995, of the total global annual input of 5,500 tons of Hg to the atmosphere from all sources, natural and anthropogenic, U.S. anthropogenic emissions contributed about 3 percent, or 158 tons. Of these, about one-third (~ 52 tons) are deposited in the lower 48 States, while the remaining two-thirds (~107 tons) diffuse beyond U.S. borders into the global reservoir. The U.S. also receives Hg deposition from the global reservoir, calculated at about 35 tons in 1995 (Ross, 1999).

The total amount of Hg emissions from coal-fired power plants is estimated to be 45 tons per year (41 metric tons) for 1999 (EPRI, 2000)). The 45 tons of Hg emissions consists of 18 tons of oxidized Hg, 26 tons of elemental Hg, and more than 1 ton of particulate Hg. The total Hg levels entering power plants in the fuel are estimated at 75 tons (68 metric tons). Therefore, the national average Hg removal is 40 percent across the existing particulate and SO_2 control technologies.

Measured removals are highly variable between the various control technology categories, as well as within some of the control technology categories.

A number of studies have been conducted on the impacts of emissions on deposition in the U.S. In general, Hg emissions from coal-fired power plants account for approximately one-third of anthropogenic emissions in the U.S. Using this ratio, an estimate of potential reductions in deposition can be obtained as a function of reductions in emissions from coal plants. Table 1 summarizes the fraction of deposition in the U.S. as estimated by different studies and the potential reduction in regional deposition that could occur.

Table 1: Reduction in U.S. mercury depositions from reductions in mercury emissions from coal-fired power plants.

Reference	% Deposition from Natural Sources	% Deposition from Global Anthropogenic Sources	% Deposition from U.S. Anthropogenic Sources	Reduction in mercury deposition assuming emissions from coal plants reduced by 50%	Reduction in mercury deposition assuming emissions from coal plants reduced by 75%	Reduction in mercury deposition assuming emissions from coal plants reduced by 90%
EPRI (2000a)	50	25	25	4.2%	6.25%	7.5%
Minnesota, (1999)	30	30	40	6.7%	10%	12%
Ross, (1999)	40*		60	10%	15%	18%
	Total mercury deposition in U.S. (tons/yr)	Coal emissions of mercury (tons/yr)	Amount of mercury deposited in U.S. from coal plants. (tons/yr)			
French (1997)	87	48	15	8.6%	12.9%	15.5%

*Ross et. al. did not differentiate between natural and global anthropogenic sources.

The estimates indicate that Hg emissions from coal are responsible for between 8.4 and 20% of the deposition in the U.S. The data reported by French, (French, 1997) specifically examined deposition of Hg from coal plants and therefore, these estimates are used as the basis for calculating risk reduction.

Data on Hg deposition from local sources are scarce. EPA states "These data are not derived from a comprehensive study for Hg around the sources of interest. Despite the obvious needs for such an effort, such a study does not appear to exist." (USEPA, 1998c, p. 3-31) EPA continues

and states “These data (Hg levels near sources) collectively indicate that Hg concentrations near these anthropogenic sources are generally elevated when compared with data collected at greater distances from the sources. However, because these data do not conclusively demonstrate or refute a connection between anthropogenic Hg emissions and elevated environmental levels, a modeling exercise was undertaken to examine further this possible connection”.(USEPA 1998c, p. 3-32). The lack of data is particularly true for deposition near coal power plants. Studies near and around coal plants (there are 3 – Four Corners, NM., Kincaid, Illinois, and Slovenia) do not conclusively show local deposition. Slight increases in sediment concentrations (20 –40%) in a nearby lake at the Kincaid plant were observed. However, increases in Hg concentration in the fish in this lake were not observed. A number of studies have shown increases in Hg concentration in soils and sediments by factors of 2 –3 within a few hundred meters of sources (Municipal waste incinerators, chlor-alkali plants, etc). The effect decreases with distance, however, several studies also show limited or no increase in Hg concentrations near sources. EPA has conducted modeling studies (EPA, 1998c) that suggest 2.5 Km downwind from a 1000 MWe coal plant, deposition could double. The modeled effects of a coal plant on deposition indicate less than a 10% increase in deposition beyond 50 Km from the plant. In this study, the local impacts of a coal-fired power plant on human health risks are not evaluated. This remains an area for future research.

5. RISK ASSESSMENT

The objective of this study is to quantify risks from Hg exposure to the fetus through consumption of fish by its mother. In addition, this study examines the reductions in risk that might occur if Hg emissions from coal-fired power plants were reduced by 50 and 90%. EPA in their report to Congress noted four regions of the country in which high deposition rates of Hg were expected, the Northeast, Southern Florida, The Great Lakes region, and the Ohio Valley region. In this report, we have combined our analysis into three regions, the Northeast, Southeast, and Midwest (a combination of Great Lakes regions and the Ohio Valley). Table 2 defines the states considered in each region when generating distributions of Hg levels in fish. In addition, particular concern is expressed for populations that consume high fractions of freshwater fish. These would include subsistence fishers (aka “fishers”) and recreational fishers. This study quantifies the risk for two subsistence fisher groups.

Table 2: States contained in the regions used in risk analysis.		
Midwest (Great Lakes and Ohio Valley)	**Northeast** (New England & Mid-Atlantic Census Division)	**Southeast** (South Atlantic and East South Census Division)
Illinois	Connecticut	Alabama
Indiana	Maine	Delaware
Michigan	Massachusetts	Florida
Minnesota	New Hampshire	Georgia
Ohio	New York	Kentucky
Wisconsin	New Jersey	Maryland
	Pennsylvania	Mississippi
	Rhode Island	North Carolina
	Vermont	South Carolina
		Tennessee
		Virginia
		West Virginia

In this study, the risk is defined as the probability of having a 5% chance of exhibiting any adverse neurological effect observed in the 3 studies used to develop the Dose Response Functions (DRF). The DRF correlates the risk with the biomarker of Hg concentration in hair, which is a function of the amount of Hg consumed through fish. The population risk is obtained through summation over all individuals that comprise the population. The population risk is then obtained from the following equation.

$$\text{Population Risk} = \Sigma_i E_i * C_i * H_i * A_i * P_i$$

Where:
i = the index for each individual.
E_i = amount of fish eaten (g/d).
C_i = Hg concentration in fish (ug Hg/g fish).
H_i = conversion factor between Hg intake (Ei*Ci ug/d) and concentration in hair (ppm).
A_i = fraction of the population that consumes E_i (g/d) of fish with a given (C_i) Hg concentration.
P_i = probability of having an adverse effect from consuming (Ei*Ci ug Hg/d) at a given hair concentration of Hg.

In practice, people consume many different types of fish and each fish has a unique concentration of Hg. Dozens of studies have been performed to characterize Hg concentrations by fish species. To account for consumption of different fish species, the above equation can be generalized as follows:

$$\text{Population Risk} = \Sigma_i (\Sigma_j E_{i,j} * C_{i,j}) * H_i * A_i * P_i$$

Where $E_{i,j}$ is the amount of fish species j consumed per day by individual i.
$C_{i,j}$ is the Hg concentration (μg/g) in fish species j consumed by individual i.

The type and amount of fish consumed as well as the amount of Hg in each fish cannot be tracked on a fish by fish basis for every individual. Thus, statistical approaches based on Monte Carlo simulation are used to estimate the fraction of population that has certain hair Hg levels. This is translated into a risk estimate by multiplying by the probability of having an effect. Each of the variables for consumption, amount of Hg in fish, and conversion of consumption to Hg level in hair are represented by a statistical distribution characterized by a mean and standard deviation. For most parameters (for example Hg content in fish and consumption rate), a log-normal distribution was found to be the most representative of the data. A normal distribution was used in the analysis for some parameters (fraction of freshwater fish consumed).

a. Population Groups Consumption

In this report, an analysis of population risks for three regions, the Northeast, Southeast, and Midwest (a combination of Great Lakes regions and the Ohio Valley) was performed. The primary risk from Hg in fish is to the fetus. Thus, the focus of the analysis was on women of child-bearing age. In different studies this is given as Age 15-44, 16-49 or other slight variations. In this report, it is assumed that all of these are the same population. The consumption of fish and shellfish (g/d) nationally was taken from month-long estimates of women age 15-44 from USEPA, 1997d. This source provided percentiles of consumption from 50 to 95%. Using successive Monte Carlo runs a log-normal distribution that matched these data was generated. These statistical parameters describing the distribution are mean = 18 g/d, standard deviation=37.3, median geometric mean =7.82, geometric standard deviation = 2.3.

There are other estimates of fish consumption that provide similar results. Jacobs et al. (1998) reported daily average per capita consumption of all finfish and shellfish by U.S. women 15-44 as 14.25 grams/person/day with 90% confidence interval of 12.96-15.55 based on cooked fish. This was based on a sample size of 2,891 from the 3-day sampling of the USDA Continuing Survey of Food Intakes by Individuals (CSFII). They report the estimated "uncooked" mean per capita consumption for the U.S. population as 20.08 g/person/day with a 90% confidence level of 18.82-21.35. They recognize that the 3-day survey is too brief to estimate an individual's usual intake.

A report by ENVIRON International Corporation (2000) reviewed consumption estimates of tuna. They report consumption of canned tuna per eating occasion for women 15-44 had a mean of 60.3 g, standard deviation 40.9 g. (lognormal approximations geometric mean 60.6 g, geometric standard deviation 40.9). ENVIRON drew frequency of consumption data from three sources. NHANES III reported times per month fish was eaten, mean = 7.5, standard deviation = 6.2. From the National Eating Trends Survey, they found a mean times per month canned tuna is eaten is 3.2, standard deviation 2.1. From the National Seafood Survey Pilot study, they found mean times per month canned tuna is eaten is 2.4, standard deviation = 2.4. These estimates provide a range of tuna consumption between 5 and 15 g/d, indicating tuna is a major fraction of fish consumption in the U.S.

The above studies incorporated both the non-fish and fish eating parts of the population. While not a national average estimate, the work of Stern et al. (1996) in New Jersey suggests that the

statistical representations above might underestimate consumption by the fraction of the population that regularly eats fish. Stern et al., (1996) estimated average consumption of fish-consuming women (18-40) in New Jersey to be a mean of 50.2 g/d and a geometric mean of 36.6 g/d.

Particular concern is expressed for populations that consume high fractions of local fish. These would include subsistence fishers and avid recreational fishers that eat the fish they catch. A detailed study of a subsistence fisher group on the Savannah River in South Carolina was taken (Burger, 1998). The consumption data from this study were used to develop a log-normal distribution that matched the data. The resulting log-normal distribution (mean 76.8 g/d, standard deviation 67.6 g/d, geometric mean 55.5 g/d, geometric standard deviation 2.17) was used as a basis for estimating consumption by subsistence fisher populations.

Table 3 summarizes the statistical parameters used for estimating consumption in this study. The data are best represented by a log-normal distribution. The table provides the mean, standard deviation, geometric mean and geometric standard deviation. In this report, the geometric mean and geometric standard deviation will be presented for all variables that are approximated with a log-normal distribution.

Table 3: Consumption statistics for population groups used in this study.

Population Group	Mean (g/d)	Standard deviation	Geometric Mean (g/d)	Geometric Standard deviation
General Population	18	37.3	7.82	2.3
Subsistence Fishers	76.8	67.6	55.5	2.17

b. Consumption by Fish Species

The general populations for the three regions consume both freshwater and marine fish. Data on fish and shellfish consumption from the U.S. Department of Agriculture's 1994-96 Continuing Survey of Food Intakes by Individuals (CSFII) was used to estimate the fraction of freshwater fish consumption for women 16-49 years of age in each region. The database contained information on fish species as well as location. The consumption by fish species was used to determine if the fish was freshwater or marine species. Entries that did not specify the fish species were ignored. For the freshwater fish consumption, it is assumed that freshwater fish were of local origin. A binomial distribution was used to estimate the standard deviation.

The fraction of marine fish is calculated such that the sum of fractions of freshwater and marine fish consumption equals 1. Table 4 presents the mean and standard deviation for freshwater fish consumption by region.

Table 4: Fraction of freshwater fish consumption.

Region	Mean	Standard Deviation
Midwest	0.24	0.039
Northeast	0.17	0.035
Southeast	0.22	0.051

The subsistence fisher populations consume only fish caught locally in this analysis. While it is probable that they consume other fish (e.g. tuna, etc.) assuming they consume only local fish maximizes the possible impact of reduction in Hg emissions. The type of local fish caught and consumed will depend on the region of the country under analysis. As an example for the Southeast, data collected by Burger (1998) on the frequency of the catch in the Southeast was used. Three fish dominated the catch, bream, catfish, and bass. The fraction of the total catch for these species was scaled to 1 ignoring the much smaller catch of shad, panfish, crappie and bowfin. The resulting distribution is:

Bream	0.52
Catfish	0.35
Bass	0.13

This distribution was used to estimate the consumption of Hg in the subsistence fisher example for the Southeast.

In the second subsistence fisher example, data on fish caught in the Savannah River (Burger, 1998 and 1999) were used to estimate consumption. The types of fish consumed and the fraction of the total are as follows;

Bream	0.64
Catfish	0.18
Bass	0.08
Others	0.10

Others include fish such as shad, panfish, crappie and bowfin.

c. Mercury Content by Fish Species

For the general population risk analyses, the Hg content in fish is needed for marine fish and for freshwater fish in the region. The U.S. average concentration of Hg in fish is a difficult issue. Many studies were designed to focus on particular pollution problems and are not representative. EPA (USEPA, 1997d, pp. 4-70 to 4-79) selected two studies they deemed to "appear to be systematic, national collections of fish pollutant concentration data." Lowe et al. (1985) found the geometric mean of all fish sampled to be 0.11 ug/g wet weight, while Bahnick et al. (1994) found a mean of 0.26 ug/g in freshwater fish.

EPA combined the Bahnick et al. (1994) data with the USDA CSFII 89-91 data. Where CSFII participants did not identify the type of fish consumed, EPA assumed the fish contained 0.26 ug/g methymercury. In part relating to the EPA default use of 0.26 ug/g, but considering the smaller value of Lowe et al. (1985), and the fact that in this study the fish consumption was representative of marine and freshwater fish, a distribution with a mean of 0.21 and standard deviation of 0.15 was used for marine fish.

To assess the Hg content in freshwater fish for each region, the database associated with "The National Survey of Mercury Concentrations in Fish, Data Base Summary 1990-1995" (www.epa.gov/ost/fish/mercurydata.htm) was used. Data were retrieved using Microsoft Access.® Queries were designed to capture the primary freshwater fish caught over an entire region (Southeast, Northeast, Midwest). The data were used to generate the statistical parameters needed in the risk assessment. All fish Hg content parameters are represented by a

log-normal distribution. Table 5 presents the mean, standard deviation, and the geometric counterparts for Hg levels in fish by region.

Table 5: Hg content (ppm) in fish by region.

	Mean (ppm)	Standard Deviation	Geometric Mean (ppm)	Geometric Standard Deviation
Marine (National)	0.21	0.15	0.17	1.90
Freshwater Midwest	0.18	0.33	0.086	3.37
Freshwater Northeast	0.39	0.82	0.17	3.67
Freshwater Southeast	0.53	0.47	0.40	2.14

In the subsistence fisher analyses, the Hg level is needed for each of the previously identified fish species (bream, catfish, and bass). For the Southeast regional example, the data associated with the "The National Survey of Mercury Concentrations in Fish, Data Base Summary 1990-1995" was used. The Hg level in the three species of fish was collected from the states that define the Southeast region, Table 2, and the statistical parameters were determined. Table 6 presents these statistical parameters.

Table 6: Statistical parameters for mercury content in fish consumed by a Southeast subsistence fisher.

Fish Species	Mean (ppm)	Standard Deviation	Geometric Mean (ppm)	Geometric Standard Deviation
Bream	0.49	0.37	0.40	1.95
Catfish	0.54	0.51	0.39	2.22
Bass	0.63	0.82	0.38	2.71

In the Savannah River subsistence fisher analyses, the database was used to determine the concentration of Hg in fish from the region of the Savannah River. Although bream was listed as the most frequent catch by fishers on the Savannah River, it did not appear in the data base. Therefore, Hg concentrations in bream from South Carolina and Georgia were used. Data and background information in this analysis was from Burger (1998), Burger et al. (2001), Burger et al. (1999), and Morris, M, Samuel, M.L (1996). The statistical parameters for the Savannah River fish are presented in Table 7. These values are approximately a factor of 2 lower than the regional values.

Table 7: Statistical parameters for mercury content in fish consumed by a Savannah River subsistence fisher.

Fish Species	Mean (ppm)	Standard Deviation	Geometric Mean (ppm)	Geometric Standard Deviation
Bream	0.25	0.18	0.20	1.91
Catfish	0.05	0.01	0.05	1.22
Bass	0.23	0.26	0.15	2.48
Other	0.14	0.10	0.11	1.90

d. Conversion of Consumption Rate to Hair Hg levels

Lipfert (1997) presented a table for correlation of Hg consumption with mean level of Hg in hair collected from 18 studies worldwide. The data were plotted on a log-log plot of consumption (ug/kg/d) vs. hair Hg (ppm) and a linear regression was performed with a best-fit slope of 0.77. This indicates that the hair Hg levels increase at a slower rate than consumption. Seventeen studies were included. The data from this report were analyzed using 17 of the studies. One study on people from Minamata, Japan was excluded as not being relevant to the current issue because concentrations of Hg were much higher than any U.S. locations. A U.S. study was a factor of 5 lower than any other study, but was retained since it was a U.S. study. The mean conversion factor from consumption (ug/d) to hair Hg (ppm) was 0.11 with a standard deviation of 0.05. To incorporate the finding that the conversion factor for consumption to hair Hg levels decreases with increasing consumption, the SPSS statistical package was used to develop the correlation (-0.516) between these values. This forced high consumption rate samples to have lower consumption to hair conversion factors in the Monte Carlo analysis.

e. Reduction in Fish Mercury

In assessing the impacts of reduction in Hg emissions from coal power plants on Hg levels in fish, we were most interested in local freshwater fish consumed by the population. Marine fish such as tuna, swordfish, shellfish, etc., will be largely unaffected by changes in U.S. emissions in Hg. This assertion is based on the fact that slightly less than 1% of the world's total Hg emissions results from coal-fired plants in the U.S. Therefore, it is likely that completely stopping Hg emissions from coal plants in the U.S. would lead to less than a 1% decrease in Hg levels in marine fish. In this study, the Hg level in marine fish is held constant.

For freshwater fish, an assumption is made that a reduction in deposition leads to a linear reduction in Hg levels in fish. The potential reductions in emission and deposition were presented in Table 1 (French, 1997 data). The data indicate that a 50% reduction in Hg emissions from coal plants would lead to an 8.6% reduction in freshwater fish Hg levels. Similarly, a 90% reduction would lead to a 15.5% reduction in freshwater fish Hg levels. These reductions are applied to the arithmetic mean Hg levels in fish (Tables 5, 6, and 7) when assessing population risks under reduced coal plant emissions scenarios.

f. Dose Response Factors: The Benchmark Dose (BMD) Approach to Risk Characterization

A review of human exposure to Hg and resulting concentrations in hair or blood is presented in Appendix A. In the U.S. the median Hg level in hair is much less than 1 ppm. Studies examining Hg levels in biomarkers (hair or blood) as a function of fish consumption do show increased median Hg levels. The increase in the median is generally less than a factor of two, however, in cases of high levels of fish consumption it can be higher. .

The basis for determining the dose response factor for Hg exposure consists of three studies conducted in the 1990's. These epidemiological studies were conducted on populations that had

high consumption of fish and therefore, high Hg levels in biomarkers. They all evaluated the impacts of Hg exposure to children and the measures of impact involved cognitive abilities (copying errors, language skills, etc). The studies, conducted in the Seychelles, Faroe Islands, and New Zealand, are discussed in detail in Appendix B.

In this report, the concept of a Benchmark Dose (BMD) is used to calculate risk. The BMD is the estimated dose corresponding to a specified incremental risk over and above background. EPA has taken the specified risk increment to be 5%. The BMD is based on a regression model of dose-response and takes into account the full range of data, not just the low end. This is the advantage of the BMD over the "no observed adverse effect level" (NOAEL) method of defining a threshold, for example. The BMD is calculated by dividing a constant by the regression slope for a given study and health endpoint. The confidence limits for the slope may then be converted into confidence limits for the BMD.

Since the BMD is a way to capture the results of each study and health endpoint in terms of a single dose value, pooling BMDs across studies and endpoints may reduce the overall uncertainty of a risk assessment. There is no universally accepted approach to pool the dose response effects from different studies. Even within a single study, there is no universally accepted way to weight different effects (NRC, 2000). In an attempt to examine a range of possible effects, three weighting approaches were used for combining the response from the three studies: straight average, average of the logarithms, and average of the reciprocals. The straight average approach tends to emphasize the data that suggest high values of hair Hg are needed to see an effect (i.e. the Seychelles study where effects were not seen), the average of the reciprocals tends to emphasize data that suggest lower values of hair Hg are indicative of an effect (New Zealand study). The latter method is consistent with the way that each BMD is derived, i.e., in terms of the reciprocal of the regression slope. Table 8 presents 16 BMD estimates that were derived from the 3 studies as reported in the recent National Research Council review report (NRC, 2000). The diversity among studies is readily apparent, and since the New Zealand Study involved fewer subjects, it was assigned a lower weight in the pooling process. The mean and standard error of each weighting process is provided in Table 9.

The pooling of the BMD results in a dose response function (DRF) that is a measure of the probability of an effect at a given exposure level. In this study, the BMD measures from the Seychelles, Faroe Islands, and New Zealand are pooled to obtain a dose response function. In the three studies, five or six possible adverse effects were evaluated and a benchmark dose was determined for each. The frequency distribution obtained by pooling BMDs and their standard errors constitutes a dose-response function, where the "response" is the probability of having a 5% chance of experiencing **any** of the various health endpoints that were pooled.

The sixteen estimates of benchmark dose from the three studies were pooled using Monte Carlo simulation to accomplish the averaging in three different ways, as shown in Table 9. Note that the mean of all three estimates is higher than the EPA "reference dose" (11 ppm). The standard errors of each of the 16 estimates were included in the simulation, and the resulting overall dose response functions (DRF) are shown in Figure 1. When a straight average is used for the 16 BMDs, a very steep DRF is obtained (right-most curve) with negligible risk below about 25 ppm hair Hg. This is consistent with the results of the Seychelles studies and with most studies on

adults. Using the reciprocal of each BMD gives a very different DRF (left-most curve) that is asymptotic to a "background" risk of about 1%, regardless of Hg exposure. The middle curve was obtained using the logarithm of each of the 16 BMDs. The notations on Figure 1 about "negative" results refer to the fractions of Monte Carlo trials that were negative. Since logarithms of negative numbers cannot be computed, 3.6% of the trials were ignored for that simulation. The (1%) negative reciprocal results were also ignored in preparing Figure 1, but the plot was extrapolated to lower hair values by assuming 1% risk at a hair concentration of 0.01 ppm. This is thus the most conservative of the three candidate DRFs. This is an arbitrary assumption meant to provide an upper bound on risks.

Figure 2 compares the slopes of the three BMD-based DRFs in terms of their relative elasticities, i.e., the relative yield in reduced risk that might be obtained from a reduction in hair Hg concentration. Only the reciprocal DRF pertains to hair Hg levels below about 5 ppm, where elasticities are quite low. At high hair Hg levels, for example 10 ppm, the reduction in risk tends to be 1:1 or higher. The steepness of the DRFs (elasticity > 1) at high Hg levels suggests that regulatory factors of safety should be applied to risk rather than to exposure.

A further aspect of MeHg epidemiology was recently introduced by Stern and Korn (2001), who argue that some portions of a population might be more sensitive than others and thus that use of a linear DRF for the whole population might obscure their responses. While no evidence was presented to support this conjecture, Monte Carlo simulations that employ disperse distributions of metabolic and pharmacokinetic parameters should be able to simulate such situations. Another way to interpret it would be as justification for confidence limits about the BMDs.

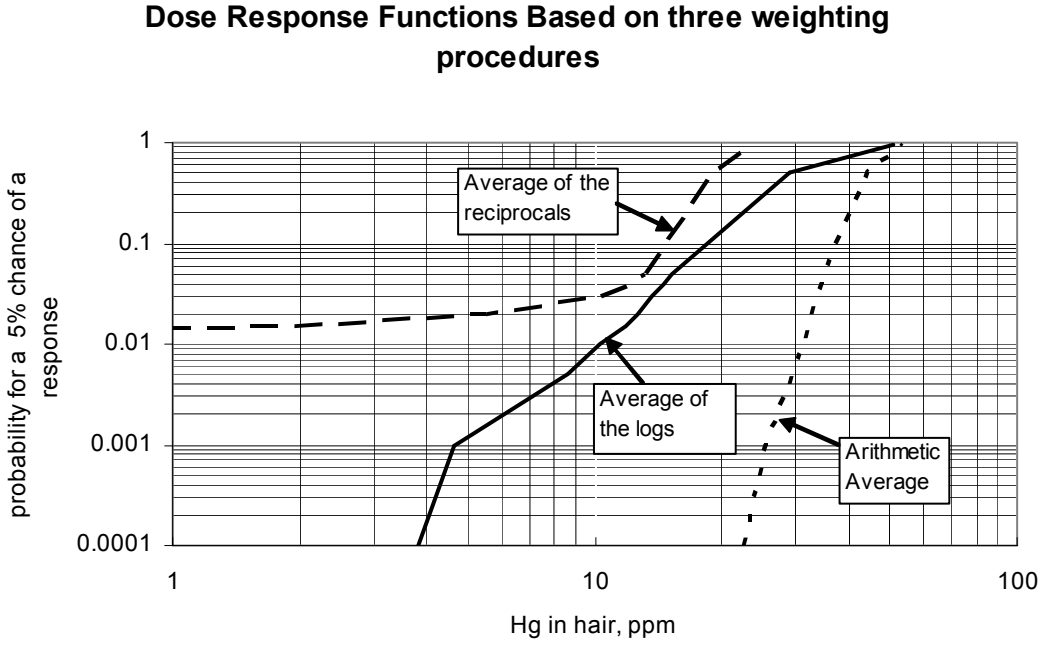

Figure 1: Pooled benchmark dose response functions for reciprocal, log, and arithmetic weighting

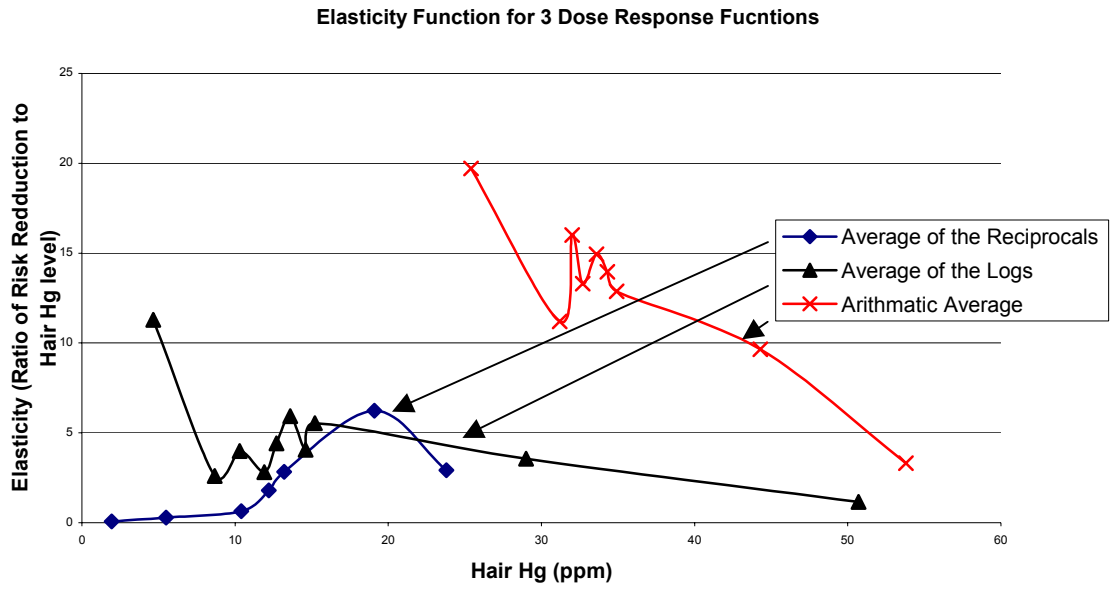

Figure 2 Elasticity (ratio of reduction in probability of effect to reduction in Hair Hg.)

Table 8 Benchmark dose estimates from the NRC Report on methylmercury.

(values expressed as ppm hair Hg [p. 284])

	BMD	**std error**
Seychelles study (weight = 1)		
Bender copying errors	100*	38.3
Child behavior checklist	21	2.0
McCarthy general cognitive	100*	39.3
Preschool language scale	100*	39.3
WJ applied problems	100*	39.8
WJ Letter/Word recognition	100*	39.8
1. = values > 100		
Faroes study (weight = 1)		
Finger tapping	20	4.1
CPT reaction time	17	3.6
Bender copying errors	28	6.6
Boston naming test	15	2.6
CVLT:delayed recall	27	6.6
New Zealand study (weight= 0.6)		
TOLD language development	12	3.1
WISC-R:PIQ	12	3.1
WISC-R:FSIQ	13	3.6
McCarthy perceptual	8	2.0
McCarthy motor test	13	3.6

Table 9. Weighted mean BMDs (ppm hair Hg).

Weight	Mean	Std error
linear	44.4	5.7
log (negatives disregarded)	28.8	2.4
reciprocal (includes negatives)	19.9	14.2

It is interesting that the largest standard error occurs for the weighting approach that uses the average of the reciprocals which has the lowest mean. This is believed to be due to the larger standard errors on the Seychelle data, even though the Seychelle data has little effect on the mean. This is an artifact of the weighting procedure and more effort needs to be performed to understand the impacts of the weighting procedure. For example, disregarding the Seychelle data for the reciprocal weighting approach, the mean would be around 15 ppm and the standard error would be around 3 – 4. This would provide a much different, and lower, estimate of risk below 10 ppm hair Hg than including the Seychelle data in the weighting. This is in spite of the fact that the Seychelle data did not see measurable effects below 10 ppm hair Hg.

The low end of the average of the reciprocals weighting DRF is an extrapolation that was used because in generating the distribution, about 1% of the hair Hg values were negative, which means that 1% of this distribution had a very low Hg value. This implies a probability of 0.01 at some undefined low Hg value which was arbitrarily selected as 0.01 ppm. Following this logic, there is a 1% chance of a risk that is effectively independent of Hg level in hair.

In this study, risk estimates were calculated based on the average of the logs weighting factor. The straight average approach indicates risk levels to the population of less than 1 in 100 million if hair Hg is less than 10 ppm. The average of the reciprocal DRF implies a background risk independent of Hg consumption in fish. To account for increases in risk due to consumption of fish contaminated with Hg would require a baseline analysis for the non-fish eating population and then an examination of incremental Hg exposure due to fish consumption. The risks could be subtracted and the impact assessed. This was not done in this study but is recommended for future work. Further discussion on the impacts of the choice of DRF on projected risk will be provided after the results of the risk estimate based on the average of the logs weighted DRF.

g. Risk Assessment Test Cases

The risk assessments performed for this analysis include 15 different test cases. For the general population 3 regions of the U.S. (Northeast, Southeast, and Midwest) were modeled for 3 Hg levels in fish (base case, 91.4% and 84.5% of the base case). For subsistence fisher populations two cases (regional Southeast fish Hg levels and Savannah River fish Hg levels) were considered for the three Hg levels at consumption rates consistent with subsistence fisher populations. Table 10 summarizes the test cases.

Table 10: Risk Assessment Test Cases.				
Population Group	Marine Fish	Local Fish	Mercury Emission Levels from Coal Plants (% of base case)	Mercury Levels in Local Fish (% of base case)
MW	Yes	Yes	Base (100%)	Base (100 %)
MW	Yes	Yes	50%	91.4%
MW	Yes	Yes	10%	84.5%
NE	Yes	Yes	Base (100%)	Base (100 %)
NE	Yes	Yes	50%	91.4%
NE	Yes	Yes	10%	84.5%
SE	Yes	Yes	Base (100%)	Base (100 %)
SE	Yes	Yes	50%	91.4%
SE	Yes	Yes	10%	84.5%
SE - Subsistence	No	Yes	Base (100%)	Base (100 %)
SE- Subsistence	No	Yes	50%	91.4%
SE- Subsistence	No	Yes	10%	84.5%
Savannah River Subsistence	No	Yes	Base (100%)	Base (100 %)
Savannah River Subsistence	No	Yes	50%	91.4%
Savannah River Subsistence	No	Yes	10%	84.5%

h. Risk Assessment Results

Monte Carlo simulations were performed that accounted for the variability in consumption, Hg levels in the different fish species consumed, and the conversion of consumption rate to Hg levels in hair for each test case. The resulting population distribution of hair Hg was used to estimate population risks using the average of the logs dose response factor. In one case, Southeast subsistence fishers, all three DRFs were used to compare risk estimates and this will be discussed separately. Table 11 presents the risk assessment results for the 15 test cases and includes the mean, geometric standard deviation, median, the population risks and the percentage reduction from the baseline case.

Median value for hair Hg ranged from 0.17 ppm in the Midwest to 0.25 ppm in the Southeast for the general population. These values are far below the EPA reference level of 11 ppm and indicate that most of the population has a very small risk from Hg exposure through fish.

The population risk of having a 5% chance of an adverse effect is quite small, ranging from 5.7 x 10^{-6} in the Midwest to 2.0 x 10^{-5} in the Southeast for the base case. The difference reflects the higher concentrations of Hg in freshwater fish in the Southeast. Table 12 contains the predicted Hair Hg level, predicted risk based on the log DRF at that hair Hg level, incremental and cumulative population risk for the Southeast base case. Examining the results of the Southeast example, it is observed that 99% of the population bears only 0.2% of the risk. The majority of

the population risk can be attributed to the 1% of the population with the highest Hg levels in hair. Similar distribution of risks applies to the two other regions.

Reducing Hg emissions from 50 – 90% over current conditions leads to a reduction in deposition of 8.6 – 15.5%. This reduction in deposition is assumed to lead to lower levels of Hg in fish. Thus, the population risks are reduced. For the general population groups, a 90% reduction in emissions led to median hair Hg levels marginally lower (< 10%) than the base case. The risk reduction was slightly larger due to the steepness of the DRFs. At the 90% reduction level, population risks ranged from 5×10^{-6} in the Midwest to 1.5×10^{-5} in the Southeast. This corresponds to a 12 to 32% reduction in risk.

To place this risk in perspective, there are approximately 4×10^{6} births/year in the U.S. (National Vital Statistics Report, 2000). Assuming that the Southeast risk level (the highest of the regions) is appropriate for the entire U.S., an estimate of 80 newborn children per year have a 5% chance of realizing any of the 16 adverse effects used to generate the DRF. If Hg emissions from power plants are reduced 90%, the number of children at risk is reduced to 60. If a more detailed analysis were performed, the estimate of the number of children at risk would decrease substantially for two reasons. First, the Southeast region has the highest risk estimates but accounts for only 24% of the births in the U.S. Second, in terms of risk reduction, approximately 12.5% of the children born in the U.S. are born in California which does not have coal-fired power plants and therefore, will not benefit substantially from reduced emissions.

Table 11 Risk assessment results.						
Population Group	Mercury Levels in Local Fish (% of base case)	Mean Hair (Hg) ppm	Geometric Standard Deviation	Median (ppm)	Population Risk	Risk Reduction from Base Case(%)
MW	Base (100 %)	0.36	3.39	0.17	5.73E-6	-------
MW	91.4%	0.35	3.42	0.16	5.39E-6	-5.9
MW	84.5%	0.34	3.49	0.16	5.01E-6	-12.6
NE	Base (100 %)	0.44	3.73	0.2	1.69E-5	-------
NE	91.4%	0.43	3.74	0.2	1.52E-5	-10.1
NE	84.5%	0.42	3.75	0.19	1.34E-5	-20.7
SE	Base (100 %)	0.49	2.33	0.25	2.06E-5	-------
SE	91.4%	0.47	2.38	0.24	1.72E-5	-16.5
SE	84.5%	0.46	2.34	0.23	1.50E-5	-32.0
SE - Subsistence	Base (100 %)	3.6	2.33	2.55	3.82E-3	-------
SE- Subsistence	91.4%	3.3	2.38	2.29	3.20E-3	-16.1
SE- Subsistence	84.5%	3.1	2.43	2.1	2.88E-3	-24.6
Savannah River Subsistence	Base (100 %)	1.41	1.99	1.12	4.35E-5	-------
Savannah River Subsistence	91.4%	1.18	2.04	0.91	2.21E-5	-49.2
Savannah River Subsistence	84.5%	1.07	2.04	0.82	1.40E-5	-67.8

Table 12 Southeast region base case general population risks.				
Predicted Hair Hg (ppm)	% Population with lower Hair Hg (ppm)	Risk at Predicted Hair Hg level	Incremental Population Risk	Cumulative Population Risk
10.3	99.9	.01	1E-5	1E-5
7.9	99.85	.0036	1.8E-6	1.18E-5
6.9	99.8	.0027	1.35E-6	1.31E-5
6.0	99.7	.0018	1.E-6	1.49E-5
5.6	99.6	.0016	1.6E-6	1.65E-5
5.2	9.5	.0014	1.4E-6	1.79E-5
4.9	99.4	.0011	1.1E-6	1.9E5
4.7	99.3	.0009	9E-7	1.99E-5
4.2	99.2	.0004	4E-7	2.03E-5
4.1	99.1	.0002	2E-7	2.05E-5
3.9	99.0	.00005	5E-8	2.06E-5
3.1	98.5	.0000045	2.25E-8	2.06E-5
2.7	98.0	.000002	1E-8	2.06E-5

There is a special concern pertaining to subsistence fishers or recreational anglers that consume large amounts of freshwater fish. These groups of people represent the high exposure cases that form the tail of the distribution of the general population. The actual risk to these groups will be highly variable and location specific. Therefore, the examples provided are intended to show the possible effects on subsistence fishers. Two cases were analyzed for the three Hg levels. One group of subsistence fishers with consumption patterns based on the studies of Burger (Burger, 1998) found along the Savannah River and fish Hg levels consistent with those in the Southeast region of the analysis. The second case used fish Hg values from fish caught in the Savannah River. Savannah River fish have substantially lower levels than the average values for the Southeast region and therefore, lower exposure to Hg. This is a consistent local impacts assessment.

Risks for the subsistence fishers using Southeast regional fish Hg levels were much higher than for the general population. Mean hair Hg levels were predicted to be 3.6 ppm, nearly a factor of 10 greater than to the general population and the population risk was $3.8\ 10^{-3}$, 2 orders of magnitude greater than to the general population. The large increase in risk reflects the non-linearity of the DRFs. The probability of observing an adverse effect increases markedly above 10 ppm Hg in hair. Approximately 5% of the subsistence fisher population is predicted to have hair Hg in excess of 10 ppm. The reduction in risk for this case due to an assumed 90% reduction in Hg was 25%, similar to the risk reduction for the general population. Figure 3 is a plot of the cumulative distribution function of the population for hair Hg for the base case and for the 90% reduction in Hg emissions for the Southeast subsistence fisher example. This example has the highest consumption rates and therefore, this shows the largest reduction from all of the test cases. The figure shows that the reductions are minor but the curve is clearly shifted to lower values.

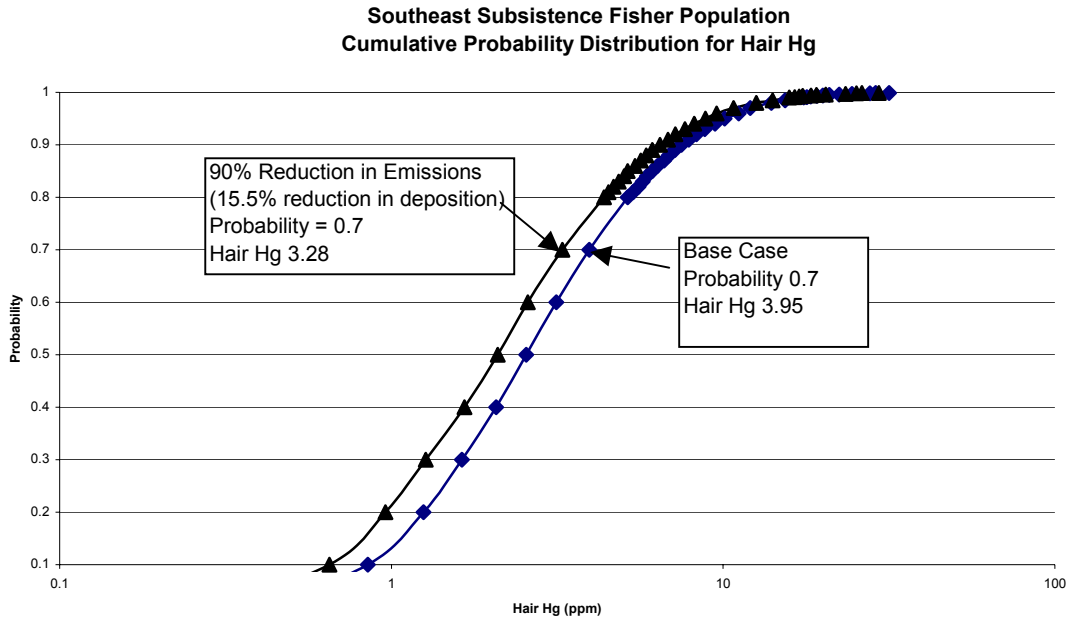

Figure 3. Predicted cumulative distribution of Hair Hg (ppm) in Southeast subsistence fisher population

As an example of the impact of using different DRFs, the Southeast subsistence fisher base case was used. Population risks for all people with more than 10 ppm Hg (lower limit for reliable reciprocal weighting probability estimates) were calculated for all three DRFs. This is the upper 5% of the population. The population risks were estimated to be 8.7 10^{-3}, 3.45 10^{-3}, and 2.0 10^{-5} for the reciprocal, log, and linear DRFs, respectively. Thus, in this region of hair Hg, the choice of the DRF can make a substantial difference in risk. It should be noted that including the entire population, the risk estimate using the log DRF was 3.82 10^{-3}. This indicates that the 95% of the population below 10 ppm bears only 10% of the total risk.

The analysis was repeated for the Southeast subsistence fisher consumption pattern, but using values of fish Hg from the Savannah River. The fish from the Savannah River had lower Hg levels than the regional average; therefore, the risks were lower than in the previous subsistence fisher case. The mean Hg level in hair for this case was 1.4 ppm, a factor of almost 3 higher than for the Southeast general population. The population risk for the Savannah River subsistence fisher was 4 10^{-5}, a factor of 2 higher than the base case and 2 orders of magnitude lower than for the Southeast subsistence fisher. This highlights the need to match local consumption patterns to local values of Hg in fish to obtain an accurate risk estimate.

6. CONCLUSIONS AND RECOMMENDATIONS FOR FUTURE WORK

EPA has stated that they are planning to regulate Hg emissions from coal-fired power plants. A quantitative risk assessment was performed to investigate the human health benefits that might occur from a reduction in Hg emissions. The primary pathway for Hg exposure is through

consumption of fish. The most susceptible population to Hg exposure is the fetus. Therefore the risk assessment focused on consumption of fish by women of child-bearing age. Dose response factors were generated from studies on loss of cognitive abilities (language skills, motor skills, etc.) by young children with mothers that consumed large amounts of fish with high Hg levels. Population risk estimates were calculated for the general population in three regions of the country, (the Midwest, Northeast, and Southeast) identified by EPA as being heavily impacted by coal emissions. Three scenarios were considered, a base case using current conditions, a 50% reduction, and a 90% reduction in Hg emissions from coal plants. These reductions in emissions were assumed to translate linearly into a reduction in fish Hg levels of 8.6% and 15.5%, respectively. Population risk estimates were also calculated for two subsistence fisher populations. These groups of people consume substantially more fish and, depending on their location, the fish may contain higher Hg levels than average. Risk estimates for these groups were calculated for the three Hg levels used for the general population analyses.

The dose response factors developed in this study are based on pooling 16 benchmark dose (BMD) estimates from three studies. The benchmark dose is the estimated dose corresponding to a 5% incremental risk of observing an adverse effect. The pooling of the BMDs results in a dose response function (DRF) that is a measure of the probability of an effect at a given exposure level (hair Hg concentration). In pooling BMDs, there is no preferred method for combining the results of different studies and the DRFs are sensitive to the pooling procedure. Therefore, three weighting procedures were used to generate DRFs, linear average, average of the logs, and average of the reciprocals, Table 9. The linear average DRF indicates that risks are very small ($< 10^{-4}$) for hair concentrations below 20 ppm. The average of the reciprocals weighting did not produce reliable results for hair concentrations below 10 ppm. For these reasons, the average of the logs DRF was primarily used in calculating population risks.

The general population risks for exposure of the in utero child to Hg are small. Estimated risks under current conditions ranged from $5.7\ 10^{-6}$ in the Midwest to $2\ 10^{-5}$ in the Southeast. Reducing emissions from coal plants by 90% reduced the estimated range in risk to $5\ 10^{-6}$ to $1.5\ 10^{-5}$ in the Midwest and Southeast, respectively. This corresponded to a risk reduction of between 12 and 32%. This is in the range of the reduction in deposition, 15.5%, however, due to the non-linear nature of the DRF, there is no direct correspondence to reduction in deposition. Examination of the distribution of population risks indicates that 99% of the population bears less than 0.2% of the total risk. The population risk, albeit small, is controlled almost exclusively by the 1% of the population with the highest hair Hg levels.

Two examples of subsistence fisher populations were simulated. Consumption for both cases was selected from data on fishers from the Savannah River. In one case, freshwater fish containing Hg levels consistent with the distribution over the entire Southeast region were used to estimate risks. In the second case, freshwater fish containing Hg levels consistent with the distribution in the Savannah River were used. The population risks for the subsistence fisher using the Southeast regional Hg levels were $3.8\ 10^{-3}$, a factor of 200 greater than the general population risk. For the subsistence fishers and the Savannah River Hg levels, the population risks were 4.3×10^{-5}, a factor of 2 greater than for the general population. Both subsistence fisher populations had the same consumption patterns, the only difference was the difference in Hg levels in the fish, which differed by approximately a factor of 2.5. The risk reduction estimated

by reducing Hg emissions from coal plants by 90% ranged from 25 to 68%, which is greater than the assumed reduction in Hg levels in fish, 15.5%.

The two order of magnitude increase in risk for approximately a factor of 2.5 higher Hg consumption rate illustrates the need to perform risk analysis tailored to local conditions when dealing with subsistence fishers. It is possible that there are small groups of subsistence fishers consuming fish with relatively high Hg content. These people may be at high risk. Another reason that local effects should be considered is that this analysis examined Hg deposition from a regional perspective. No attempt was made to perform local deposition modeling immediately downwind from coal plants. Local deposition could lead to small regions with fish Hg levels considerably higher than the values used in this report. If this is the case, small subsistence fisher populations might have some risk from Hg. For these reasons, it is recommended that future work involve examination of local deposition around coal plants using plant emission data, and local data on waterways, population, and fish Hg concentrations that could be impacted.

To examine the effects of the choice of Dose Response Factor on predicted risk, the population risk for the subsistence fisher in the Southeast using the regional Hg levels in fish was calculated for all three DRFs. The predicted risks were estimated to be $8.7\ 10^{-3}$, $3.45\ 10^{-3}$, and $2.0\ 10^{-5}$ for the reciprocal, log, and linear DRFs, respectively. The choice of DRF makes a substantial impact on predicted risk in this case. This highlights the need for future work to improve the DRFs through examination of new data as it becomes available and reanalysis of existing data.

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Appendix A Human Exposure to MeHg and Concomitant Health Effects

Diet is the most important source of MeHg, and seafood appears to be the most important dietary component. However, as discussed below, analysis of human biomarkers suggests that there may also be sources other than seafood. Many studies have shown that the developing fetus is the most susceptible human receptor; MeHg passes through the placenta and through the brain-blood barrier. There is also limited direct evidence (from autopsies) of a strong correlation between MeHg in infant brains and in maternal hair and blood (Cernichiari et al., 1995).

However, there is perhaps less agreement on which of the available biomarkers constitutes the best surrogate measure of fetal exposure. Maternal hair levels comprise a record of Hg intake during the entire pregnancy, but the most critical period of exposure during fetal brain development has not been established. By contrast, the Hg present in umbilical cord tissue or blood best represents maternal Hg intake shortly before delivery. Maternal blood Hg content may be influenced by transient exposure to MeHg shortly before sampling. The utility of all such biomarkers is compromised by individual variability in the ratios among them and, implicitly, their relationships with fetal brain Hg levels. Such variability comprises a source of "measurement error" that can attenuate the statistical relationships derived from epidemiology (Lipfert, 1997).

Epidemiology studies comprise the only source of direct data on human dose-response relationships for methylmercury, although such findings are generally also supported by animal toxicology data. These epidemiology studies differ in important ways with respect to most other such studies intended to quantify the health effects of air pollution. For example, epidemiology studies involving the inhalation pathway use concentrations in ambient air, as routinely measured at central monitoring stations, as the "dose" measure for all subjects living in the community in question. This assumption entails major statistical uncertainties due to individual variability in the actual exposures to specific constituents of the urban air pollution mixture. By contrast, MeHg epidemiology studies work with data on individual exposures. In general, biomarker concentrations are preferable to estimates of dietary intake, which are subject to the vagaries of uncertain recall and of lack of data on the exact Hg contents involved. By and large then, data on fish consumption are more useful as classification variables than they are as surrogate exposure variables.

A.1 A Simplified Risk Assessment Paradigm

The population risk of a specified health effect may be estimated as the sum of the products of the incremental probability of exposure at a given level for each member of the population times the probability of experiencing the effect at that exposure level. Information on such responses are typically obtained from a "dose-response" function, where some measure of individual exposure serves as a proxy for the dose to the target organ, here the developing fetal brain.

This paradigm requires data on the distribution of exposures (either measured or calculated) and a dose-response function, both expressed in terms of the same exposure metric. Typically, human Hg exposures are expressed as concentrations in hair or in blood, although concentrations

in umbilical cords have also been used. If only data on dietary intakes are available, then a pharmacodynamic model should be used to estimate the resulting steady-state body burdens.

Calculated exposures should account for variability in two populations: (1) the human population, each of which consumes fish to a varying extent and varies in terms of Hg retention time; (2) the fish population being sampled, which varies in Hg content according to species and size

Exposure data are most reliable at the low exposures typically experienced by most people. However, the reliability of population exposure estimates also depends on the fraction of samples that are below the lower detection limit of the analytical methods used. Thus, a reliable frequency distribution requires methods sufficiently precise to capture the low values and a population sufficiently large to include a representative number of higher exposures.

Response data are typically most reliable at the highest exposures where unambiguous responses are most likely. The population must be large enough to have a reasonable chance of detecting infrequent responses at lower exposure levels. Extrapolations below the lower limit of the data are entirely determined by the choice of the mathematical dose-response function model. Although highly exposed individuals may be postulated and may even exist, population data are required to place such risks in a public health context.

A.2 Data on Human Exposures

For this report, we drew on published information on biomarker concentrations, mainly Hg concentrations in hair and in blood. Such information is subject to the Hg species measured (total Hg vs. MeHg), the population demography (age, race, gender), locations, dietary habits, and access to seafood. An open question is whether, after considering these modifying factors, human exposures have been changing over time.

Table A-1 presents data on Hg concentrations in human hair. Median or geometric mean concentrations are presented as a measure of central tendency instead of arithmetic means because of the typically log-normal frequency distributions. For the United States, medians in ostensibly non fish-eating groups range from below detection limits (New Jersey females) to 0.24 ppm in a national sample of females from the 1980s. For populations that eat fish, median levels range from about the same to about 50% higher, suggesting that the additional exposure to MeHg in the U.S. from eating fish is quite modest. However, the contributions of seafood to Hg in foreign population is much higher, with median concentrations up to an order of magnitude higher than in the U.S. and with an approximate doubling due to fish consumption for those studies that made the distinction. Median hair Hg concentrations tend to be higher in populations that engage in sport fishing, presumably because of greater fish consumption. The data in Table A-1 do not show consistent differences between total Hg and MeHg reporting. With respect to frequency distributions, geometric standard deviations (GSDs) range from 2.3 to 2.6 except for the NHANES IV data, which are substantially more disperse at 3.1.

Comparable data on Hg in blood are given in Table A-2. The “standard” ratio of hair concentration to blood concentration is about 250, so that 1 ppm in hair corresponds to about 4

ppb in blood. However, these ratios are quite variable, as may be seen by comparing data in Tables A-1 and A-2 for those studies that reported both measures. Differences may also be expected in terms of the relative contributions of inorganic Hg in hair and in blood. Again, the U.S data compare reasonably well and are bracketed by Canadian data on people who fish recreationally. Florida Native Americans are in this same range, but the highest levels by far are those reported by Wheatley and Paradis (1995) for Canadian native peoples living in the Arctic. However, those levels appear to have declined substantially since that sampling program began in the early 1970s (Figure A-1, from Wheatley and Paradis).

Frequency distributions of these biomarker data are presented in Figures A-2 to A-4. These plots include measured data (discrete data points) and extrapolated trends, based on the overall GSD implied for each dataset. Hair Hg data are shown in Figure A-2 and show that exposures greater than the EPA reference dose (equivalent to 11 ppm) are quite rare. The highest concentrations are those obtained by extrapolating the NHANES IV data (well beyond the actual limit of the data, it should be noted), because of the extraordinarily high GSD of those data. The sponsoring agency, the U.S. Centers for Disease Control, was unwilling to release the actual data to us, thus precluding an examination of the reasons for the high GSD. The increment in the probability of experiencing a hair Hg level over about 1 ppm due to eating fish is about a factor of 3, which is small compared to the overall envelope of exposure uncertainty (the spread between the dashed lines). Exposure uncertainties increase greatly for hair Hg levels greater than about 0.5 ppm. Note that levels tend to be somewhat lower in EPA Region 5, which is where much of the nation's coal combustion takes place. The use of total Hg rather than MeHg in that data accentuates this apparent deficit.

Frequency distributions of the blood Hg data are shown in Figures A-3 and A-4. Figure A-3 shows data from NHANES IV, New Jersey, and Quebec; again, a very large range is seen with the NHANES data standing out as especially high and the New Jersey data for pregnant females as low. Of course, it is possible that pregnant females deliberately limit their intake of contaminated fish, but in any event, those data represent their actual exposures. Note that all of the data in Figure A-3 were obtained in recent years, which was not the case for Figure A-2 (the Smith et al. data are from the early 1980s). Figure A-4 shows the Canadian arctic data and links it with the extrapolated NHANES distribution. Note the large difference in GSDs; it is difficult to believe that NHANES actually includes any data as high as 100 ppb for persons within the contiguous United States, whereas many such data points were obtained previously in northern Canada.

Because of these important uncertainties in the NHANES IV data, we decided not to consider those data further for this assessment, pending an opportunity to examine the actual dataset in its entirety.

TABLE A1 Hg EXPOSURE AS MEASURED IN HAIR

median or geometric mean hair levels, ppm (GSD)

author	population	w/o fish	w/fish	% increment
Smith et al.	U.S. females (MeHg)	0.24 (2.6)	0.36 (2.5)	50
Stern et al.	NJ preg. fem. (MeHg)	~0	0.36	?
Pellizari et al.	EPA Region V (total Hg)		0.20	
Fischer et al.	Michigan pregnant fishers (total Hg)		0.52	
NHANES IV	U.S. females (total Hg)		0.20 (3.1)	
Lee et al.	Korean females (MeHg)	0.51	3.7	625
Kosatsky et al	Montreal fishers (total Hg)	0.38 (2.3)	0.82 (2.5)	116
Hacon et al.	Amazon preg. fem. (total Hg)	0.52	0.93	79
Santos et al.	Brazil, ages 16-40 (total Hg)		4.36	
	(no gold mining, 13 fish meals/wk)			
Batista et al.	Spanish children (total Hg)	0.45	0.78	73
Soria et al.	Spanish pregnant females (MeHg)		0.70	
Feng et al.	Chinese males, 40-49 (MeHg)	0.37		
	Japanese males, 40-49 (MeHg)		4.7	
Grandjean et al.	Faeroe Island mothers (fish+whale)	0.84	4.5	436
	(fish only)		2.3	174

TABLE A2 Hg EXPOSURE AS MEASURED IN BLOOD
median or geometric mean blood levels, ppb (GSD)

author	population	w/o fish	w/fish	% increment
NHANES IV	U.S. females (total Hg)		1.2 (3.1)	
Stern et al.	NJ pregnant females (total Hg)		< 1.0	
Anderson	Great Lakes fishers (total Hg)		3.4	
	Florida Native Americans (total Hg)		2.3	
Mahaffey	Quebec fishers (MeHg)	0.50	0.7	40
Kosatsky	Montreal fishers (total Hg)	1.44	3.03	
Seifert et al.	German adults (total Hg)	0.30	0.77	156
Bergdahl	Swedish females (1968 MeHg)	2.0		180
	(1981 MeHg)		2.0	
Soria	Spanish pregnant females (MeHg)		3.0	
Rhainds	Quebec cord blood (total Hg)	3.0		310
Svensson	? (total Hg)	1.8		
Vahter	Swedish pregnant females (MeHg)		0.73	
Schweinsberg	German fishers (total Hg)	0.6	2.41	350
Trepka	German children (total Hg, polluted area)	0.25		
Grandjean et al.	Faeroe Island mothers (fish+whale)	3.1	24.2	680
	(fish only)		10.8	248
Wheatley	Canadian Indians (MeHg)		25 (1.9)	

Appendix B Epidemiology Studies

In our FY2000 effort, we reviewed epidemiology studies on the health effects of Hg that were published since 1996, together with a few earlier studies that were deemed important for context.

B.1 Adult Epidemiology Studies

Four recent epidemiology studies of adults were reviewed, covering populations in the Amazon basin, near Minamata Bay, Japan, in a gold mining area of Tanzania, and of Cree Indians in northern Quebec. The Amazon study involved 91adults with hair Hg < 50 ppm. Minor neurological symptoms were seen, and there may have been a threshold in these responses around 15 ppm. Gold miners and fishermen in Tanzania had total hair Hg concentrations up to 950 ppm, but with the exception of 14 subjects, mean total Hg was < 10 ppm. The authors were unable to relate symptoms to hair Hg levels, and it appeared that persons with < 5 ppm were free of symptoms related to Hg poisoning. The Cree Indian subjects, who had been monitored for about 25 years, were tested with a new apparatus for rapid alternating movements. There were significant performance differences between 6 subjects with mean hair Hg = 27.5 ppm and 6 age-matched controls with mean hair Hg of 8.5 ppm. These three studies thus suggest that the threshold for onset of minor neurological symptoms may lie somewhere in the range from 5 to 15 ppm total Hg in hair.

The fourth study was concerned with possible long-term adverse effects from previous high-level exposures to MeHg stemming from the Minamata Bay disaster. This population consumed fish frequently (an average of 2-3 oz/d) and had correspondingly high blood Hg levels (20-28 ppb in red blood cells). However, it was not possible to distinguish whether any long-term effects should be attributed to current or to the much higher previous exposures.

B.2 Epidemiology Studies of Children

Ten papers and subsequent correspondence were reviewed that reported various aspects of epidemiology studies on children. Five of these were concerned with the Faroes Islands study, three with the Seychelles Islands study, and one with children who live downstream from gold mining operations in the Amazon basin. Exposures in all three situations were much higher than those experienced in the United States.

The Amazon study (Grandjean et al., 1999) involved 352 children in 4 different communities. Most children ate fish twice a day, and the "low" exposure community had a geometric mean total hair Hg concentration of 3.8 ppm, which is an order of magnitude higher than median levels seen in the United States. The results of the neurological tests were inconsistent, especially when compared on a village basis, leading to the (alternative) conclusion that a threshold may exist somewhere between 12 and 18 ppm in hair total Hg. However, inorganic Hg (IHg) is an issue around gold mining operations and it is not clear how much of the hair Hg was present as MeHg or as inorganic Hg.

The four Seychelles papers were largely consistent in reporting no adverse effects in the main Seychelles cohort (n=738). The 1998 paper by Axtell et al. found no relationship between prenatal exposures to MeHg (median maternal hair total Hg = 5.8 ppm) and the age at which children first talked, and very slight delays (ca.1 day) in the age at first walking. This cohort was very precocious, with mean ages at walking and talking of 10.5-11 mos. The results of a large battery of neurological tests at 66 months of age were published in JAMA in 1998 (Davidson et

al.); no adverse results were reported and some of the findings were positive (i.e., Hg was beneficial). This paper generated substantial debate by means of an accompanying editorial and the subsequent correspondence and rejoinders. As a result of some of the ensuing criticism of the Seychelles study, an additional brief paper was published in JAMA (Cox et al., 1999) that addressed the question of separate vs. joint regression against prenatal and postnatal exposure indices. Since the two exposures were poorly correlated, it made no difference. The final Seychelles paper that was reviewed (Crump et al., 2000) was intended to develop a benchmark dose (see below for definition and discussion) from the various analyses that have been done at different ages of the cohorts. The average of the benchmarks was 25 ppm, a value that is lower than all but one of the measured prenatal exposures. This benchmark analysis is thus tantamount to finding no adverse effects.

Findings from the five Faroes papers that were reviewed are somewhat more diverse. The most important paper (aside from the 1992 exposure paper discussed above) is probably the 1997 paper by Grandjean et al., published in Neurotoxicology and Teratology. The Faroes cohort is slightly larger than the Seychelles cohort, has alternative exposure metrics, was evaluated less frequently, and is influenced by two different sources of MeHg: fish, mainly cod fish, and whale, which also contains high levels of PCBs. A large battery of tests was administered to the children at age 7 y; Hg-related dysfunctions were found in several areas of performance. These results were robust to the inclusion of confounders and to the deletion of highly exposed children (total hair Hg > 10 ppm). However, the models used were based only on log-transformed concentrations of total Hg in umbilical cord blood. A subgroup whose mothers ate no whale meat was identified in the 1992 exposure analysis paper; this group should have been used as controls and evaluated separately in order to establish the (claimed) absence of a threshold. The next paper (Grandjean et al., 1998) used a case-control approach, in which hair Hg was used to define the two groups. By comparing these subgroup medians with the data tabulated in the 1992 exposure paper, the diversity among these alternative exposure measures becomes apparent. The influence of whale consumption was apparent by comparing the exposure statistics of the two groups: the mothers of cases ate 50% more fish meals per week (3 vs.2), 200% more whale meals per month (3 vs.1), had almost 5 times the cord blood Hg and almost 7 times the hair Hg and 80% more PCBs in the umbilical cords. It seems clear that any neurological performance differences between cases and controls (which were very slight) should be attributed to eating whale (and the various contaminants therein). It is unfortunate that this hypothesis was not tested directly.

In 1999, Grandjean et al. published a comparison of exposure biomarkers with regard to their associations with test results at birth and at ages 1 and 7. However, PCB concentrations were not among them, and results were given only for log-transformed measures. These results were variable, and not all of them agreed with the previous papers. Since none of the exposures captured either the mean or peak exposures during pregnancy and since only logarithmic models were used, substantial questions remained. The final Faroes neurological paper reviewed here was a benchmark dose analysis published in Toxicology Letters (Budtz-Jorgensen et al., 2000). The alternatives considered were: cord blood vs. maternal hair Hg, linear vs. logarithmic, and either fifth or tenth percentiles considered as "abnormal," leading to eight sets of results. The use of cord blood Hg vs. hair Hg made little difference when standard errors were considered, especially for the linear model. The benchmark doses ranged from 11.5 to 26 ppm (based on converting the cord blood results to equivalent hair units), in conformance with the studies discussed above based on other cohorts. However, when the logarithmic transform was used, the benchmark doses were much lower, from 1.3 to 15.6 ppm. Thus, the use of the logarithmic transform appears to be a critical feature of the Faroes studies.

The final Faroes paper that was reviewed considered the hypothesis that MeHg is an adverse cardiovascular risk factor, as measured in this case by blood pressure and heart rate differences in the 7-year old children. Again, the shapes of the dose-response functions are key, in that associations were only found in the lower range of exposures, in contrast to some of the supporting studies that were cited. Again, no effort was made to distinguish fish-only diets from fish+whale diets, and the possibilities of vitamin C or other dietary deficiencies were not considered.